

## Clinical Image

Open Access, Volume 6

# Bright fornix in Wernicke's encephalopathy

Somarajan Anandan<sup>1\*</sup>; Caroline Surendran Pushpalettha<sup>2</sup>

<sup>1</sup>Department of Neurology, St Joseph Hospital, Anchal, Kollam, Kerala, India.

<sup>2</sup>Department of Medicine, St Joseph Hospital, Anchal, Kerala, India.

**\*Corresponding Author: Somarajan Anandan**

Department of Neurology, St Joseph Hospital,  
Anchal, Kerala, India.

Email: drsomarajan@yahoo.co.in

Received: Feb 17, 2025

Accepted: Mar 12, 2025

Published: Mar 19, 2025

Archived: www.jcimcr.org

Copyright: © Anandan S (2025).

DOI: www.doi.org/10.52768/2766-7820/3516

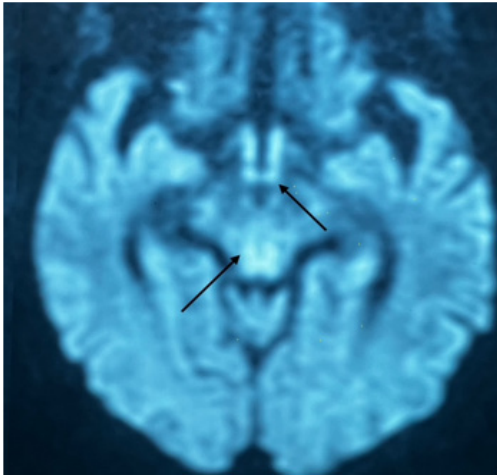
### Description

A 70-year-old man with parkinsonism presented with altered sensorium of 3 days duration. He had a history of fever and cough one week back and was admitted in local hospital and had several intravenous fluid infusions. There was no history of alcohol intake. His only medication was levodopa/carbidopa. He has a history of poor food intake since several weeks. On examination he was drowsy and had bilateral external ophthalmoplegia and gaze evoked nystagmus. There was no limb weakness or limb incoordination. Magnetic resonance imaging of brain showed hyperintensity of bilateral paramedian thalamus, mamillary body, periaqueductal region and pons. In addition, fornix showed hyperintensity in diffusion weighted image and fluid attenuated inversion recovery images (Bright Fornix) (Figures 1-3). There was no diffusion restriction. Wernicke's encephalopathy is an acute neuropsychiatric disorder resulting from thiamine deficiency and is characterized by a clinical triad of confusion, ocular abnormalities, and ataxia. Only 16% of patients show full clinical triad and 19% of patients have none of these signs [1]. Thiamine deficiency causes dysfunction of the Krebs cycle and the pentose phosphate pathway. Magnesium is a necessary cofactor for the conversion of thiamine into its active form, Thiamine Diphosphate (TDP). Magnetic Resonance Imaging (MRI) typically shows bilaterally symmetrical lesions

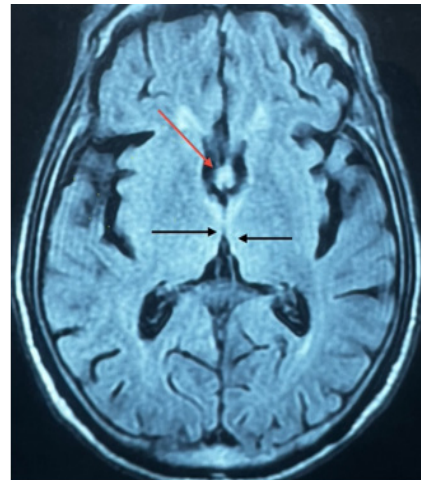
in the paraventricular regions of the thalamus, hypo thalamus, mammillary bodies, periaqueductal region and floor of the fourth ventricle. MRI may also show unusual sites of brain lesions such as the putamen, caudate, splenium of the corpus callosum, dorsal medulla, pons, red nucleus, substantia nigra of the midbrain, cranial nerve nucleus, vermis, dentate nucleus, paravermian region of the cerebellum, fornix and pre- and post-central gyri [2]. Involvement of white matter tracts such fornix is only rarely reported [3].

### References

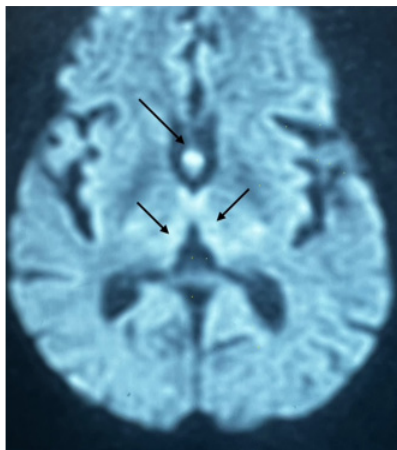
1. Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: A retrospective analysis of 131 cases diagnosed at necropsy. *J Neurol Neurosurg Psychiatry*. 1986; 49(4): 341-5. doi: 10.1136/jnnp.49.4.341.
2. Zuccoli G, Pipitone N. Neuroimaging findings in acute Wernicke's encephalopathy: Review of the literature. *AJR Am J Roentgenol*. 2009; 192: 501-8. 10.2214/AJR.07.3959.
3. RS Borges, N Ventura, EL Gasparetto, MVR Pinto. Forniceal Involvement in Wernicke Encephalopathy *American Journal of Neuroradiology*. 2011; 32 (11): 209. DOI: 10.3174/ajnr. A2888.



**Figure 1:** Axial diffusion weighted image of brain showing hyperintensity of periaqueductal region, mamillary bodies and hypothalamus.



**Figure 3:** Axial fluid attenuated inversion recovery image brain showing hyperintensity of bilateral paramedian thalamus and fornix.



**Figure 2:** Axial diffusion weighted image of brain showing bilateral symmetric paramedian thalamic hyperintensity and hyperintensity of fornix (Bright fornix).